Continuing Professional Development

MODULE 9: JUNE 2011

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We are confident that this CPD Section of the Irish Journal of Psychological Medicine will prove to be a valuable resource for consultant psychiatrists, psychiatric trainees and all journal readers. We welcome feedback from readers and, especially, any suggestions for topics to be covered in future CPD modules. Suggestions should be emailed to: psychological@medmedia.ie

Serotonin toxicity

Sobia Nasim, Faraz Jabbar, Asfar Afridi, Brendan D Kelly

Serotonin toxicity is a potentially life-threatening condition associated with a range of psychotropic medications, co-administration of specific combinations of agents and overdose of certain drugs. It is associated with a wide diversity of clinical signs and symptoms, including cognitive, autonomic and somatic effects, as well as serious complications, including possible death. Diagnosis is often challenging and requires a high index of suspicion. Differential diagnosis includes syndromes such as neuroleptic malignant syndrome. Management depends on the causal agent and urgency of clinical presentation. Treatment may involve discontinuing the causal agent and providing supportive measures, or emergency intervention to preserve vital functions (airway, breathing, circulation), amongst other measures. Further research is needed to clarify the incidence of serotonin toxicity, issues related to differential diagnosis, optimal management of the condition, and treatment of mood problems following serotonin toxicity.

Introduction

Owing to increased use of serotonergic agents throughout psychiatry, serotonin toxicity has become increasingly relevant to clinical practice.[1] Serotonin toxicity came to widespread attention over two decades ago when an eighteen-year old patient in New York died following co-administration of meperidine and phenelzine.[2] Since then, this serious adverse effect has been referred to variously as serotonin toxicity, serotonin syndrome, serotonin storm, serotonin toxicity syndrome and hyper-serotonaemia. Regardless of the phrase used, serotonin toxicity is a potentially life-threatening drug reaction that may occur in a range of situations including therapeutic drug use, deliberate self poisoning and co-administration of certain agents or classes of agents.[2]

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Epidemiology of serotonin toxicity

Despite the common use of serotonergic medications, it appears likely that serotonin toxicity is under-diagnosed, owing, at least in part, to its occasionally subtle presentation.[3] Epidemiological data are, therefore, limited, although one English study identified 0.4 cases of serotonin toxicity per 1000 patient-months of treatment with nefazodone.[4] In addition, however, some 85% of general practitioners were unaware of serotonin toxicity.

In 2002, the Toxic Exposure Surveillance System, which receives case descriptions from office-based practices, inpatient settings, and emergency departments, reported 26,733 incidences of exposure to selective-serotonin reuptake inhibitors that caused significant toxic effects in 7,349 persons, resulting in ninety-three deaths.[2,4] Overall, severe cases of serotonin syndrome carry a mortality rate between 0.1% and 12%.

Several different pharmacological mechanisms can cause excess of serotonin (5-hydroxytryptamine [5-HT]) in the central nervous system; these include:

- Inhibition of the metabolism of serotonin (monoamine oxidase inhibitors).
- Prevention of the reuptake of serotonin in nerve terminals (serotonin reuptake inhibitors).
- Increased serotonin precursors (tryptophan).
- Serotonin release (serotonin-releasing agents).[5]

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Table 1: Clinical features and complications of serotonin toxicity

Cognitive effects	Autonomic effects	Somatic effects	Complications
Restlessness	Shivering	Tremor	Metabolic acidosis
Anxiety	Flushing	Shivering	Rhabdomyolysis
Agitation	Sweating	Hypertonia	Seizure
Easily startled	Tachycardia	Rigidity (lower limb greater than upper)	Renal failure
Confusion	Mydriasis	Akathesia	Disseminated intravascular coagulopathy (secondary to hyperthermia)
Hypomania	Diaphoresis	Hyperreflexia	
Hallucinations	Hypertension	Myoclonus	
Delirium	Hyperthermia (mild<38.5C, severe>38.5C)	Ocular clonus (slow continuous lateral eye movements)	
Coma	Diarrhea (with hyperactive bowel sounds)	Inducible clonus	
		Spontaneous clonus	

Note: These clinical features and complications of serotonin toxicity are derived from various diverse sources in the literature.[2,5-7]

The resulting excess serotonin acts on various serotonin receptors (5HT1A, 5HT1D, 5HT2A, 5HT2B, 5HT3, 5HT4) to produce the clinical features of serotonin toxicity. The main features linked to various receptors include:

- Nausea, vomiting, anxiety and gastrointestinal motility, mediated by 5HT3 and 5HT4 receptors
- Stomach contraction, mediated by 5HT2B receptors
- Muscle tone and locomotion, mediated by 5HT1D receptors
- Neuronal excitation, learning, peripheral vasoconstriction, platelet aggregation, mediated by 5HT2A receptors
- Neuronal inhibition, regulation of sleep, feeding, thermoregulation, hyperactivity associated with anxiety, hypoactivity associated with depression, mediated by 5HT1A receptors.[2,6]

Clinical presentation

The clinical features of serotonin toxicity include a broad range of cognitive, autonomic and somatic effects (*see Table 1*).[2,5-7] Serotonin toxicity can constitute an acute medical emergency, commonly precipitated by co-administration of a combination of psychotropic agents, such as a monoamine-oxidase inhibitor and selective-serotonin reuptake inhibitor.[8] A serotonin toxicity scale has also been devised by Hegerl et al for individuals with depression being treated with paroxetine[5,9] although this is not in widespread clinical use.

Radomski et al suggest a need for sub-classification of this condition and divided it into mild serotonin symptoms, serotonin syndrome, and toxic states.[10] Similarly, Isbister et al divide the spectrum of serotonin toxicity into three levels of severity:

- **Mild serotonergic symptoms:** These may or may not interfere with the patient and may occur with therapeutic use of many serotonergic agents.
- **Moderate toxicity:** Supportive and symptomatic treatment is needed as the symptoms cause the patient significant distress.
- **Severe serotonin toxicity or serotonin crisis:** This is a medical emergency and is caused by combination of drugs acting on various sites most commonly including a monoamine-oxidase inhibitor and selective-serotonin reuptake inhibitor. It is characterized by a rapidly increasing temperature associated with muscle rigidity, and will progress to multi-organ failure if not treated within hours.[1,5,8]

Medications associated with serotonin toxicity

All medications that affect the steps in serotonin metabolism or regulation can potentially cause serotonin toxicity. Specific examples cited in the literature, either alone or in combination, include selective-serotonin reuptake inhibitors[11,12] such as paroxetine;[13] monoamine-oxidase inhibitors[14] such as linezolid[15,16] and selegiline;[17] tricyclic antidepressants[18] such as clomipramine[19] and imipramine;[20] selective-noradrenaline reuptake inhibitors[21] such as venlafaxine;[22-24] St John's wort;[25,26] buproprion;[27] and L-tryptophan[15,28] (*see Table 2*). Other medications associated with serotonin toxicity, either alone or in combination, include metoclopramide;[15] amphetamines;[29] opioid analgesics[21,30] such as tramadol[13] and meperidine;[31] fentanyl;[32] methadone;[33,34] dextromethorphan;[35] lithium;[22,23] and certain triptans.[18]

This list is not necessarily exhaustive, however, and is subject to change. Other agents which have been linked to serotonin toxicity (alone or in combination) include, for example, certain anti-histamines[18] and certain anti-emetics.[11] Readers are advised to consult the most recent literature for further information on these and other relevant agents and combinations.

The clinical literature on serotonin toxicity is based chiefly on case-reports and case-series, so it is exceedingly difficult to determine specific risk-factors associated with mild, moderate and severe serotonin toxicity. It appears, nonetheless, that comorbidity may increase risk of serious morbidity: venlafaxine, for example, has been associated with serotonin syndrome causing severe rhabdomyolysis and acute renal failure in a patient with idiopathic Parkinson's disease.[24]

It is similarly difficult to determine which medications or other risk factors are more likely to be associated with fatal as opposed to non-fatal serotonin toxicity. Overall, however, the literature, such as it is, suggests that fatal serotonin toxicity may be most commonly associated with certain combinations of agents, such as imipramine and tranylcypromine[20] or fluoxetine and selegiline.[17] Further research and surveillance are needed to clarify this further.

Diagnostic difficulties

Serotonin toxicity can be notably difficult to diagnose. In 1991, Sternbach developed diagnostic criteria for serotonin toxicity,[36] but the clinical features now associated with serotonin toxicity are presented and categorized in *Table 1*.[2,5-7] As the symptoms may be of a generally non-specific nature, serotonin toxicity may be, in certain cases, a diagnosis of exclusion, especially when a known serotonergic drug was recently added or increased in dose. As a result of these diagnostic challenges, serotonin toxicity may be missed completely or misdiagnosed, as many symptoms overlap with conditions such as neuroleptic malignant syndrome.[5] Serotonin toxicity symptoms may also overlap with normally occurring drug responses (eg. hypertension, tachycardia and hyperthermia with the use of methylenedioxymethamphetamine), and this is another factor which may contribute to under-reporting of serotonin toxicity.

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Against this background, Dunkley et al conducted a retrospective study and developed another diagnostic approach, based on the Hunter serotonin toxicity criteria.[37] According to this approach, in the presence of serotonergic agents within the previous five weeks, the following symptoms are considered diagnostic of Hunter serotonin toxicity:

- Spontaneous clonus
- Inducible clonus and either agitation or diaphoresis
- Ocular clonus and agitation or diaphoresis
- Tremor and hyper-reflexia
- Hypertonia and temperature > 38°C, with ocular clonus or inducible clonus.

From a clinical perspective, it is especially important to make distinction between serotonin toxicity and neuroleptic malignant syndrome, in order to guide appropriate intervention.[11] The distinguishing features of neuroleptic malignant syndrome include the absence of neuromuscular excitation and presence of bradykinesia, lead-pipe rigidity and extra pyramidal symptoms. Neuromuscular excitation will be absent in central nervous system infection, encephalitis, meningitis, anticholinergic delirium, sympathomimetic toxicity and malignant hyperthermia. Moreover, in anti-cholinergic delirium there is dryness of the skin and absence of bowel sounds. Other differential diagnoses include non-convulsive seizures (in which an electroencephalogram is helpful) and acute baclofen withdrawal (where history and response to baclofen may help in differentiation).[5,38]

An important differential diagnosis is fibromyalgia. One case-report details a patient with serotonin toxicity who was misdiagnosed with fibromyalgia due to presence of diffuse soft tissue pain.[39] This patient also, however, exhibited other significant symptoms including agitation, change in mental status, diarrhoea, myoclonus, hyperreflexia and poor coordination. This case illustrates the importance of careful history taking and relevant investigations when diagnosing or excluding serotonin toxicity or related diagnoses.

Management of serotonin toxicity

There are currently no randomised, placebo-controlled trials to guide pharmacological treatment of serotonin toxicity, although case reports and series suggest several medications may be beneficial in certain cases.[6] The intensity of therapy depends upon the severity of symptoms and urgency of the clinical presentation:

- Mild serotonin symptoms respond well to discontinuation of offending agents, supportive measures and waiting for symptoms to resolve.
- Moderate cases should have all cardiovascular and thermal abnormalities corrected and there may be a role for serotonin antagonists such as cyproheptadine.[40,41] Oral cyproheptadine (4-12mg) is probably the most useful antagonist and has the occasionally beneficial side-effect of sedation.
- Severe cases of serotonin toxicity are medical emergencies, requiring attention to airway, breathing and circulation. Cooling of the patient, sedation and muscle paralysis may be more important than pharmacological treatment.[5] Since cyproheptidine is unlikely to be effective in patients administered activated charcoal, chlorpromazine or olanzapine can be used, under certain circumstances, if indicated.[5,42]

Caution is needed in the use of antipsychotics in this patient group as not only can neuroleptic malignant syndrome be easily misdiagnosed as serotonin toxicity, but some case reports have implicated antipsychotics as precipitants of serotonin toxicity in individuals receiving serotonergic drugs.[43,44] Extreme caution is advised.

Table 2: Medications associated with serotonin syndrome (alone or in combination)

- Selective-serotonin reuptake inhibitors[11,12] such as paroxetine[13]
- Monoamine-oxidase inhibitors[14] such as linezolid[15,16] and selegiline[17]
- Tricyclic antidepressants[18] such as clomipramine[19] and imipramine[20]
- Selective-noradrenaline reuptake inhibitors[21] such as venlafaxine[22-24]
- St John's wort[25,26]
- Buproprion[27]
- L-tryptophan[15,28]
- Metoclopramide[15]
- Amphetamines[29]
- Opioid analgesics[21,30] such as tramadol[13] and meperidine[31]
- Fentanyl[32]
- Methadone[33,34]
- Dextromethorphan[35]
- Lithium[22,23]
- Triptans[18]

Note: This list is not necessarily exhaustive as the list of relevant agents is subject to change. Other agents which have been linked to serotonin toxicity (alone or in combination) include, for example, certain anti-histamines[18] and certain anti-emetics.[11] Readers are advised to consult the most recent literature for further information on these and other relevant agents and combinations.

Management of mood problems in individuals with depression who develop serotonin toxicity can be especially challenging. There is a notable paucity of literature to provide guidance on risk of serotonin toxicity under such circumstances. However, recommencement after adequate washout period should not necessarily present an increase in risk.[6] It would be advisable to use less serotonergic agents and assess patients weekly for several weeks after initiating treatment, according to the guidelines on depression by the Canadian Coalition for Seniors' Mental Health.[45] Again, extreme caution and clinical surveillance are recommended, along with increased research into this topic.

Conclusions

With increased use of serotonergic medications, clinical syndromes resulting from serotonin excess, which can result in serotonin syndrome, may become increasingly common.[39,46] Serotonin toxicity can result from drug overdose, drug interactions or adverse effects involving serotonergic or other agents. Selective-serotonin reuptake inhibitors are among the common drugs taken in overdose and may result in symptoms of toxicity in up to 15% of cases of overdose.[47] Most life-threatening reactions are related to drug-drug interactions which may involve common over-the-counter medications. In addition, drugs such as fluoxetine which have long half lives can interact with other medication up to five weeks after discontinuation.[5,48] Against this background, a high index of suspicion for serotonin toxicity is needed in all patients showing autonomic or mental status changes and neurological findings, following administration of relevant agents.

Further research is needed in order to clarify the precise nature and incidence of adverse-effects of drugs affecting serotonin

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levels, differential diagnosis of resultant clinical syndromes, optimal management of serotonin toxicity, and issues related to recommencement of antidepressants and management of mood problems following serotonin toxicity. For the moment, detailed history taking, careful examination and investigation, and maintaining a high index of suspicion will result in optimal diagnosis and management of serotonin toxicity at present levels of knowledge.

Declaration of Interest: None.

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Multiple Choice Questions: Module 9

1. Serotonin syndrome is:

Commonly over-diagnosed	T	F
Well-recognised amongst clinicians	T	F
An important clinical syndrome	T	F
Never fatal	T	F
Only seen in men	T	F

2. Pharmacological mechanisms of serotonin toxicity include:

Inhibition of serotonin metabolism	T	F
Prevention of serotonin reuptake	T	F
Increased serotonin precursors	T	F
Reduced dopamine	T	F
Increased serotonin release	T	F

3. Recognised consequences of excess serotonin can include:

Nausea and vomiting	T	F
Stalibization of mood	T	F
Stomach contraction	T	F
Improvements in neurological function	T	F
Neuronal inhibition	T	F

4. Clinical features of serotonin toxicity include:

Reduction in mood symptoms	T	F
Restlessness	T	F
Disseminated intravascular coagulopathy	T	F
Tachycardia	T	F
Elimination of neurological deficits	T	F

5. Treatment of serotonin toxicity may include:

Discontinuing the offending agent	T	F
Increasing serotonin levels	T	F
Serotonin antagonists	T	F
Cooling the patient	T	F
Emergency attention to airway, breathing and circulation	T	F

Answers on www.ijpm.org

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